



International Journal of Surgery Case Reports

journal homepage: www.casereports.com

A case of intensive care unit-acquired weakness after emergency surgery for acute abdomen

Tetsuro Tominaga^a, Takashi Nonaka^a, Hiroaki Takeshita^a, Yuichiro Honda^b, Hiroki Nagura^b, Toshio Shiraishi^a, Masaki Kunizaki^a, Yoriyuma Sumida^a, Shigekazu Hidaka^a, Terumitsu Sawai^{c,*}, Takeshi Nagayasu^a

^a Departments of Surgery, Cardiopulmonary Rehabilitation Science, Division of Surgical Oncology, Japan^b Departments of Surgery, Rehabilitation, and Cardiopulmonary Rehabilitation Science, Division of Surgical Oncology, Japan^c Cardiopulmonary Rehabilitation Science, Division of Surgical Oncology, Japan

ARTICLE INFO

Article history:

Received 11 March 2016

Received in revised form 16 May 2016

Accepted 22 May 2016

Available online 24 May 2016

Keywords:

ICUAW

Critical illness

Rehabilitation

Surgeon

ABSTRACT

INTRODUCTION: Surgeons often perform surgery for patients who are critically ill. Intensive care unit (ICU)-acquired weakness (ICUAW) is a condition in which systemic and prolonged muscle weakness occurs and causes worse short-term and long-term outcomes.

PRESENTATION OF CASE: A 60-year-old woman with sudden nausea and vomiting presented to our hospital and developed shock. Abdominal CT showed thickness of the descending colon and ascites. She was diagnosed with sepsis due to descending colon cancer. Colectomy of the descending colon was performed due to necrosis, and a stoma was created. After surgery, she received intensive care in the ICU. It was difficult to wean her from the ventilator, and she developed severe flaccid weakness of the limbs. Her Medical Research Council (MRC) sum score was 0. Since other organic disorders causing prolonged paralysis were excluded, she was finally diagnosed as having ICUAW. Active rehabilitation, nutritional support, and glycemic control were continued. Now, 6 months after surgery, her MRC score has improved to 30.

DISCUSSION: The treatment for ICUAW has been reported to include recovery from the critical illness, early rehabilitation, and nutritional support, and it requires close cooperation among health care providers.

CONCLUSION: All physicians and surgeons who care for critically ill patients should take active steps to diagnose ICUAW in order to avoid deterioration of patients' activities of daily living.

© 2016 The Author(s). Published by Elsevier Ltd on behalf of IJS Publishing Group Ltd. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

1. Introduction

It has been reported that marked loss of muscle strength can occur in patients with life-threatening infections [1], and this may also cause an ongoing disorder of function and loss of QOL after discharge from hospital [2]. Intensive care unit-acquired weakness (ICUAW) is such a neuromuscular disorder caused by severe illness treated in the ICU [3]. ICUAW has been reported to occur in various

patients, and early rehabilitation is considered very important to prevent prolonged muscle weakness [4,5]. However general surgeons still seem to be insufficiently aware of this clinical condition. A case of ICUAW after severe sepsis is presented.

2. Presentation of case

A 60-year-old woman with sudden nausea and vomiting presented to our hospital. She was on steroid treatment for malignant lymphoma. She had pain in the left side of the abdomen, and a large mass was palpable. There was no muscular guarding or rebound tenderness. Laboratory data showed increased inflammation and marked acidosis (Table 1). Abdominal CT showed thickness of the descending colon, swelling of surrounding lymph nodes, and ascites (Fig. 1a, b). Her blood pressure and level of consciousness decreased gradually. She was diagnosed with sepsis due to descending colon cancer. Colectomy of the descending colon was performed due to necrosis, and a stoma was created (Fig. 1c). After surgery, the patient was admitted to the intensive care unit for general care. In the ICU, she received not only treatment for the primary disease,

Abbreviations: ICUAW, ICU-acquired weakness; MRC, Medical Research Council.

* Correspondence to: Department of Cardiopulmonary Rehabilitation Science, Nagasaki University Graduate School of Biological Sciences, 1-7-1 Sakamoto, Nagasaki 852-8501, Japan.

E-mail addresses: tomitetsu2000@yahoo.co.jp (T. Tominaga), tnonaka@nagasaki-mc.com (T. Nonaka), takehiro@nagasaki-u.ac.jp (H. Takeshita), yhonda@nagasaki-u.ac.jp (Y. Honda), hnagura@nagasaki-u.ac.jp (H. Nagura), tshiraishi@nagasaki-u.ac.jp (T. Shiraishi), kunizaki@nagasaki-u.ac.jp (M. Kunizaki), y-sumida@nagasaki-u.ac.jp (Y. Sumida), hidaka-s@nagasaki-u.ac.jp (S. Hidaka), sawai@nagasaki-u.ac.jp (T. Sawai), nagayasu@nagasaki-u.ac.jp (T. Nagayasu).

<http://dx.doi.org/10.1016/j.ijscr.2016.05.038>

2210-2612/© 2016 The Author(s). Published by Elsevier Ltd on behalf of IJS Publishing Group Ltd. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

Table 1
Laboratory data before surgery.

Peripheral blood	
RBC	$5.54 \times 10^4/\text{mm}^3$
Hg	12.7 g/dl
Hct	43.1%
WBC	$50800/\text{mm}^3$
Plt	$8.0 \times 10^4/\text{mm}^3$
Biochemistry	
T-Bil	0.4 mg/dl
AST	106 IU/l
ALT	71 U/l
LDH	571 IU/l
ALP	155 U/l
CK	139 IU/l
AMY 82	82 mg/dl
Na	135 mg/dl
K	5.3 mg/dl
Cl	120 mg/dl
BUN	34 mg/dl
Cr	2.74 mg/dl
TP	5.4 g/dl
Alb	3.8 g/dl
Procalcitonin	0.635 ng/ml
Coagulation system	
PT(%)	17%
PT-INR	4.15
APTT	unmeasurable
AT-III	21%
FDP	44.6 $\mu\text{g/ml}$
D-dimer	18.2 $\mu\text{g/ml}$
Blood sugar	
BS	145 mg/dl
Blood gases (O ₂ 10l)	
pH	6.889
PO ₂	455 mmHg
PCO ₂	26.1 mmHg
HCO ₃	−4.7 mmol/l
BE	−28.2 mmol/l
Lactate	13.2 mmol/l

but also early rehabilitation to prevent muscle atrophy. Though she recovered from multiple organ failure, she had difficulty in being weaned from the ventilator, and a tracheotomy was performed on postoperative day 12. Her level of consciousness was GCS4 (E4VTM0). There was no anisocoria, abnormal light reflex, disorder of eye movement, or facial muscle paralysis. She then developed severe flaccid weakness of the limbs and diminished deep tendon reflexes. Her Medical Research Council (MRC) sum score was 0. Head CT showed no abnormalities causing muscle weakness (Fig. 2). She was diagnosed as having ICUAW from the clinical course and the pathophysiological findings, including the MRC score. Active rehabilitation, nutritional support, and glycemic control were continued. Now, 6 months after surgery, her MRC score has improved to 50 (Fig. 3).

3. Discussion

The mortality rate of sepsis in the ICU has improved due to the development of intensive treatment. Currently, not only short-term outcomes, but also long-terms outcomes are being considered during critical care.

In the nineteenth century, loss of flesh and strength in patients with life-threatening disease was reported [6]. In 2009, Stevens and colleagues reported a framework for diagnosing and classifying ICUAW [7]. The diagnostic criteria for ICUAW are practical, based on history and clinical examination: (1) generalized weakness developing after the onset of critical illness; (2) weakness is diffuse (involving both proximal and distal muscles), symmetric, flaccid, and generally spares cranial nerves; (3) MRC sum score <48, or mean MRC score <4 in all testable muscle groups noted on more than two occasions separated by >24 h; (4) dependence on mechanical ventilation; and (5) causes of weakness not related to the underlying critical illness excluded. To make a definitive diagnosis of ICUAW, costly or invasive tests such as electromyography, single-nerve condition studies, muscle biopsy, and muscle ultrasound are required. However, obtaining complete data from

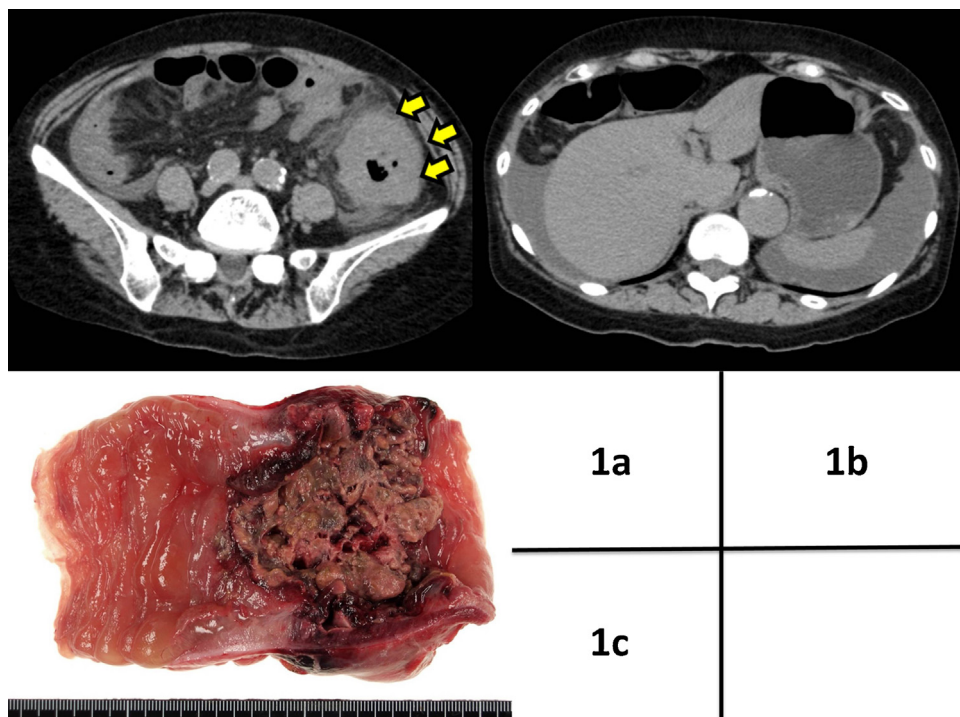


Fig. 1. Abdominal CT on admission and excised specimen findings. Abdominal CT shows thickness of the descending colon, swelling of surrounding lymph nodes, and ascites (a, b). A large tumor with necrosis is located in the descending colon. Lymph nodes surrounding the tumor show bulky swelling (c).



Fig. 2. Head CT. Head CT shows no abnormalities causing muscle weakness.

critically ill patients at the bedside remains challenging because of tissue edema and electrical interference [8]. The current patient presented with systemic weakness that spared the cranial nerves after emergency surgery for acute abdomen. She had an MRC sum score of 0 and could not be weaned off mechanical ventilation. Since various examinations could not identify an organic cause for paralysis, a presumptive diagnosis of ICUAW was made.

It has been reported that about 26–65% patients who require mechanical ventilation have weakness on awakening [4,5], and the longer the patients are ventilated, the higher the incidence of muscle weakness [9].

The clinical condition and pathogenesis of ICUAW have not been fully determined. However, it has been reported that the humoral immune reaction due to a severe condition such as septic shock, multiple organ failure, and acute respiratory distress syndrome

plays an important role in the development of ICUAW [4]. The production of various types of cytokines leads to degeneration of nerve conduction, microvascular hyperpermeability due to degeneration of microvessels, and production of neurotoxic agents. In addition, muscle breakdown is directly affected by metabolic disease, inflammatory changes, and loss of energy [10]. In ICUAW patients, not only skeletal muscle, but also the diaphragm can be affected [11]. Thus, such patients usually fail weaning off mechanical ventilation that cannot be explained by their respiratory and circulatory status.

ICUAW is often characterized by symmetrical weakness of the limbs that affects proximal muscles more than distal muscles [12]. The function of cranial nerves is usually unaffected [13]. Thus, patients with ICUAW typically grimace with painful stimuli but show no withdrawal of the limbs. The present patient showed a prolonged need for mechanical ventilation despite improvement of her general condition. In addition, she showed a discrepancy between the facial muscles and core muscles, as discussed above.

ICUAW is associated with delayed weaning from ventilation, longer ICU stay, and increased ICU/hospital mortality [14]. In a recent study, patients with weakness showed a decline over time from 36% at hospital discharge to 22% at 3 months, 7–15% at 6 months, 4–14% at 1 year, and 9% at 2 years [15]. In general, septic patients with multiple organ failure have a high mortality, up to 60% [16]. Among the survivors, patients whose general condition improves in the relatively early phase tend to avoid worsening of ADL on a long-term basis [4]. Thus, the main risk factor for ICUAW appears to be the severity of illness. Therefore, early and rigorous intensive care is crucial to decrease mortality and improve long-term outcomes [17]. Another study reported that sarcopenia, use of vasopressors, corticosteroids, neuromuscular blockage, and aminoglycoside therapy, immobilization, and hyperglycemia were independent risk factors for neuromuscular complications [8,18]. In the present case, the patient had extremely severe septic shock and required prolonged use of vasopressors, and her muscle weakness did not recover for a long time.

There are no specific therapeutic options for preventing or treating ICUAW. Risk factor avoidance or modification might reduce the severity and risk of ICUAW. One aspect of treatment for ICUAW is prevention of contractures and pressure ulcers by early rehabilitation and frequent position changes [19]. Malnutrition was originally considered a contributor to ICUAW, so early tube feeding should be considered [20].

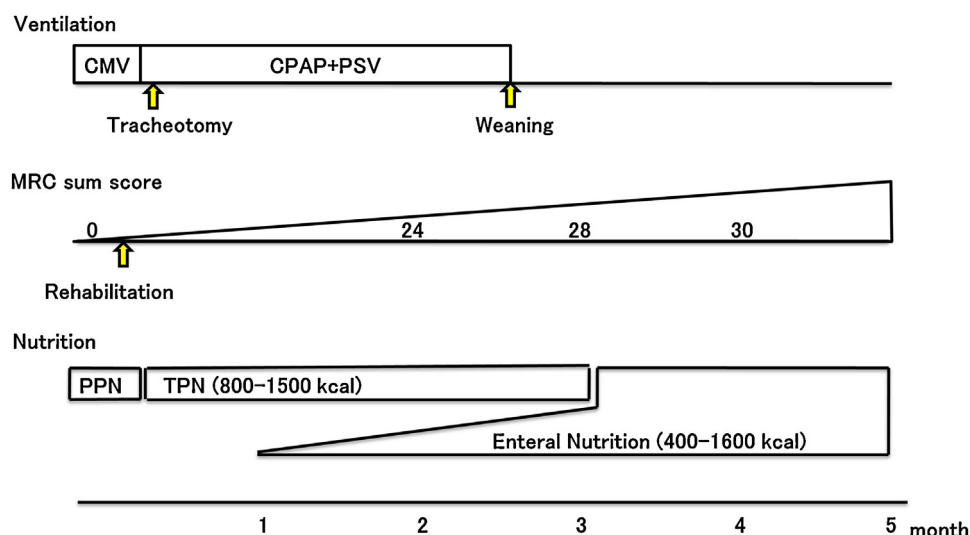


Fig. 3. Clinical course. MRC: Medical Research Council, CMV: continuous mandatory ventilation, CPAP + PSV: continuous positive airway pressure + pressure support ventilation, PPN: peripheral parenteral nutrition, TPN: total parenteral nutrition.

Jolley and colleague reported that critical illness can be categorized into four phases: pre-ICU, early ICU, late ICU, and post ICU [8]. They suggested that assessment and interventions targeting individualized stages could be an effective strategy.

The concept of ICUAW has been generally recognized among intensive care clinicians, and many reports have been published in the field of critical care. However, few studies have been reported in the field of general surgery [21]. Thus, it is important to collaborate with healthcare providers such as nurses, nutritional support teams, and physical therapists to prevent the development of ICUAW. ICUAW should be diagnosed earlier, and appropriate prevention and management could prevent a decrease in long-term ADL.

4. Conclusion

Surgeons commonly operate on patients with critical illnesses, and ICUAW is not rare condition in such patients. All physicians who care for critically ill patients should take active steps to diagnose ICUAW in order to avoid decreases in patients' ADL.

Conflict of interest

None.

Funding

This study was not supported by any grant.

Ethical approval

Not applicable.

Consent

Written and signed consent from the patient to publish a case report has been obtained.

Author contributions

Tetsuro Tominaga, Takashi Nonaka, Hiroaki Takeshita, and Toshio Shiraishi developed the study concept. Yuichiro Honda and Hiroki Nagura performed rehabilitation. Masaki Kunizaki, Yori-hisa Sumida, and Shigekazu Hidaka collaborated in medical care. Terumitsu Sawai and Takeshi Nagayasu were involved in manuscript revision.

Guarantor

Terumitsu Sawai.

References

- [1] M.S. Herridge, A.M. Cheung, C.M. Tansey, A. Matte-Martyn, N. Diaz-Granados, F. Al-Saizi, et al., Canadian critical care trials group: one year outcomes in survivors of acute respiratory distress syndrome, *N. Engl. J. Med.* 348 (2003) 683–693.
- [2] M.S. Herridge, C.M. Tansey, A. Matte, G. Tomlinson, N. Diaz-Granados, A. Cooper, et al., Canadian critical care trials group: functional disability 5 years after acute respiratory distress syndrome, *N. Engl. J. Med.* 364 (2011) 1293–1304.
- [3] G. Hermans, G.V. Berghe, Clinical review: intensive care unit acquired weakness, *Crit. Care* 19 (2015) 274–282.
- [4] N.A. Ali, J.M. O'Brien, S.P. Hoffmann, G. Phillips, A. Garland, J.C. Finley, Acquired weakness, handgrip strength, and mortality in critical ill patients, *Am. J. Respir. Crit. Care Med.* 178 (2008) 261–268.
- [5] T. Sharshar, G.S. Bastuji, R.D. Stevens, M.C. Durand, I. Malossin, P. Rodriguez, Presence and severity of intensive care unit acquired paresis at time of awakening are associated with increased intensive care unit and hospital mortality, *Crit. Care Med.* 37 (2009) 3047–3053.
- [6] W. Osler, *Principles and practice of medicine* 1st ed. New York, Appleton, 1892.
- [7] R.D. Stevens, S.A. Marshall, D.R. Cornblath, A.H. Hoke, D.M. Needham, B. Jonghe, et al., A framework for diagnosing and classifying intensive care unit-acquired weakness, *Crit. Care Med.* 37 (2009) 299–308.
- [8] S.E. Jolley, A. Bunnell, C.L. Hough, Intensive care unit acquired weakness, *Chest* 3692 (2016) 47575–47576.
- [9] H. Mizakhani, J.N. Williams, J. Mello, S. Joseph, M.U. Meyer, K. Waak, Muscle weakness predicts pharyngeal dysfunction and symptomatic aspiration in long-term ventilated patients, *Anesthesiology* 119 (2013) 389–397.
- [10] H. Fink, M. Helming, C. Unterbuchner, A. Lenz, F. Neff, J.A. Martyn, et al., Systemic inflammatory respiratory syndrome increases immobility-induced neuromuscular weakness, *Crit. Care Med.* 36 (2008) 910–916.
- [11] R.A. Shanely, M.A. Zergeroglu, S.L. Lennon, T. Sugiura, T. Yimlamai, D. Enns, et al., Mechanical ventilation-induced diaphragmatic atrophy is associated with oxidative injury and increased proteolytic activity, *Am. J. Respir. Crit. Care Med.* 166 (2002) 1369–1374.
- [12] B. De Jonghe, T. Sharshar, J.P. Lefaucheur, F.J. Authier, I. Durand-Zaleski, M. Boussarsar, Paresis acquired in the intensive care unit: a prospective multicenter study, *J. Am. Med. Assoc.* 288 (2002) 2859–2867.
- [13] C.F. Bolton, D.A. Lavery, J.D. Brown, N.J. Witt, A.F. Hahn, W.J. Sibbald, Critically ill polyneuropathy: electrophysiological studies and differentiation from Guillain-Barre syndrome, *J. Neurol. Neurosurg. Psychiatry* 49 (1986) 563–573.
- [14] E. Fan, D.W. Dowdy, E. Colantuoni, P.A. Mendez-Tellez, J.E. Sevransky, C. Shanholtz, Physical complications in acute lung injury survivors: a 2-year longitudinal prospective study, *Crit. Care Med.* 42 (2013) 849–859.
- [15] D.M. Needham, V.D. Dingas, P.E. Morris, J.C. Jackson, C.L. Hough, P.A. Mendez-Telles, Physical and cognitive performance of patients with acute lung injury 1 year after initial trophic versus full enteral feeding. EDEN trial follow-up, *Am. J. Respir. Crit. Care Med.* 188 (2013) 567–576.
- [16] L. Manship, R.D. McMillan, J.J. Brown, The influence of sepsis and multisystem and organ failure on mortality in the surgical intensive care unit, *Am. Surg.* 50 (1984) 94–101.
- [17] S. Nanas, K. Kritikos, E. Angelopoulos, A. Siafaka, S. Tsikriki, M. Poriazi, Predisposing factors for critical illness polyneuromyopathy in a multidisciplinary intensive care unit, *Acta Neurol. Scand.* 118 (2008) 175–181.
- [18] F.S. Leijten, J.E. Harinck-de Weerd, D.C.J. Poortvliet, The role of polyneuropathy in motor convalescence after prolonged mechanical ventilation, *J. Am. Med. Assoc.* 274 (1995) 1221–1225.
- [19] K. Berek, Polyneuropathies in critically ill patients: a prospective evaluation, *Intensive Care Med.* 22 (1996) 849–855.
- [20] C.F. Olton, G.B. Young, Critical illness polyneuropathy due to parenteral nutrition, *Intensive Care Med.* 23 (1997) 924–925.
- [21] T. Tominaga, H. Wada, K. Tou, S. Shibasaki, T. Oka, A case of critical illness polyneuropathy developed after surgery, *J. Jpn. Surg. Assoc.* 72 (2011) 1616–1621.

Open Access

This article is published Open Access at [sciencedirect.com](https://www.sciencedirect.com). It is distributed under the [IJSCR Supplemental terms and conditions](#), which permits unrestricted non commercial use, distribution, and reproduction in any medium, provided the original authors and source are credited.